APPLICATION OF HEALTH INFORMATION TO HAZARDOUS AIR POLLUTANTS MODELED IN EPA'S CUMULATIVE EXPOSURE PROJECT[4]

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Relatively little is known about the spectrum of health effects, and the scope and level of ambient air concentrations of those pollutants regulated under the Clean Air Act as "hazardous air pollutants." The U.S. Environmental Protection Agency's (USEPA) Cumulative Exposure Project uses currently available emissions inventories, from a variety of source types, and an atmospheric dispersion model to provide estimates of ambient concentrations for 148 hazardous air pollutants (HAPs) in over 60,000 census tracts for the year 1990. This paper uses currently available hazard information for those pollutants and provides a database of potential regulatory threshold concentrations

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2. Abbreviations: ATSDR, Agency for Toxic Substances and Disease Registry; CAA, Clean Air Act and amendments of 1990; CAL/EPA, California Environmental Protection Agency; CERCLA, Comprehensive Environmental Response Compensation and Liability Act; ED10, effective dose for a 10 percent cancer response above background; EPA, United States Environmental Protection Agency; GWP, pollutant identified as being of concern in the Great Waters report to Congress; HAPs, Hazardous Air pollutant as defined in the Clean Air Act of 1990; HCP, pollutant identified for severe toxicity from relatively low long- or short-term exposure in the section 112(g) hazard ranking; IARC, International Agency for Research on Cancer; IDLHs, immediately dangerous to life and health levels; IUR, Inhalation Unit Risk; LOC, Level of Concern; MRLs, Minimal Risk Levels; NO_x , nitrogen oxides; NTP , National Toxicology Program; RELs, Reference Exposure Levels; RfC, inhalation reference concentration; RfC_{dt}, inhalation reference concentration for developmental toxicity; RfD, reference dose; SARA, Superfund Admendments and Reauthorization Act; SO_2, sulfur dioxides.

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of concern, or "benchmark concentrations," and a methodology for prioritizing and characterizing the quality of the data. In order to demonstrate application of the database and prioritization scheme to outputs from the Cumulative Exposure Project, comparisons were made with the maximum modeled concentration of each individual hazardous air pollutant across the census tracts. Of the 197 benchmark concentrations for cancer and non-cancer (long- and short-term exposures) effects compiled for the study, approximately one half were exceeded with a predominance of exceedance of cancer benchmarks. While the number of benchmark concentrations available to fully characterize potential health effects of these pollutants was limited (approximately 80 percent of HAPs identified as cancer concerns had benchmark concentrations for cancer and 50 percent of all HAPs had non-cancer benchmark concentrations) and there was greater uncertainty in derivation of maximum modeled air concentrations than other levels, the comparison between the two was a useful approach for providing an indication of public health concern from hazardous air pollutants.

INTRODUCTION

The Clean Air Act (CAA) as amended in 1990, defines two classes of pollutants: criteria pollutants and hazardous air pollutants [HAPs, also know as "air toxics" (Stern, 1992)]. The criteria pollutants — lead, NO_x, SO_2, particulate matter, ozone, and carbon monoxide— are regulated by standards that govern their ambient levels for the whole country and are based on an extensive database of health and welfare information (U.S. Government, 1990). In addition, monitoring networks are set up across the country to ascertain whether areas are in compliance with the standards. Consequently, the information on health effects and ambient concentrations of the criteria pollutants is relatively rich (Woodruff et al., 1997).

In contrast, little information is available on health effects and outdoor concentrations of HAPs (USEPA, 1994b,c,d; Woodruff et al., 1997, 1998). These pollutants are listed in section 112(b)(1) of the CAA and include 189 specific pollutants or chemical groups. Currently, the lack of information on ambient concentrations of HAPs across the country hinders efforts to assess potential health effects and prioritize and evaluate policy initiatives for reduction of ambient levels. The large number of pollutants to track, the varied nature of the HAPs (e.g., chemistry, half-life, and toxicity), and the potential heterogeneity in distribution and magnitude of concentrations make monitoring a large number of HAPs over a large area infeasible (Kelly et al., 1994; Woodruff et al., 1997).

The air toxics component of the U.S. Environmental Protection Agency's (USEPA) Cumulative Exposure Project has been designed to provide estimates of outdoor air concentrations for a large portion of the HAPs (148 pollutants) and information on populations affected in individual census tracts (Woodruff et al., 1997; Rosenbaum et al., 1998). The project uses existing atmospheric modeling methods and emissions data for multiple sources to estimate long-term average HAP concentrations for the year 1990. Model estimates were developed for each of the 60,803 census tract in the continental U.S. and represent contributions from both stationary and mobile sources. The modeled concentrations approximate the population weighted average of outdoor HAP concentrations experienced within a census tract over the course of a year (Rosenbaum et al., 1998).

This paper describes the development of a database and methodology for application of health hazard information to such estimates of ambient concentration. The methodology used currently available characterizations of the health hazard of these pollutants to determine levels that represent potential regulatory thresholds of concern or "benchmark concentrations." To demonstrate the application of the set of benchmark concentrations to the Cumulative Exposure Project model outputs, benchmark concentrations were compared to the highest modeled census tract concentration for each HAP, thus providing the number of benchmark concentrations exceeded in at least one census tract. Identification of modeled HAP concentrations greater than these benchmark concentrations serves as a first step in the identification of those communities which may merit further study, or specific pollutants that may be of most concern.

MATERIALS AND METHODS

Benchmark concentrations and qualitative health effects information for 148 HAPs were assembled from a variety of sources, evaluated comparatively, and then assigned to a series of tiers defined by the quality, availability, and consistency of derivation. Much of the needed information and science policy judgments were previously compiled for USEPA's proposed rulemaking under section 112(g) of the CAA and were supplemented by information from several other data sources as described below (Caldwell-Kenkel et al., 1993, 1995; Shoaf et al., 1994; USEPA, 1994 a,b,c,e).

This analysis used three types of benchmark concentrations representing carcinogenic hazard, and short- and long-term non-carcinogenic hazard. Accordingly, the first goal of the analysis was to collect quantitative dose-response information for as many health endpoints and from as many comparable methods as available for these three hazard categories.

For each hazard category, benchmark concentrations representing a presumptive health protective level was selected. For carcinogenic hazard, the benchmark was selected to be the concentration of a known, probable, or possible human carcinogen representing the upper bound of a one in a million excess probability of contracting cancer over a lifetime of exposure. This benchmark was based on provisions of CAA sections 112(f) and 112(c)(9) that allow source categories to be exempted from regulation and residual risk to be negligible when posing less than a one in a million lifetime risk to the most exposed individual (U.S. Government, 1990).

For non-carcinogenic hazards, benchmarks were selected to be the concentration of a HAP likely to be without appreciable risk of non-cancer effects from long- or short-term exposures. The language in the CAA in sections 112(f) and 112(c)(9) describes such levels to be that needed to protect public health with an "ample margin of safety" (U.S. Government, 1990). The Inhalation Reference Concentration (RfC) has been used in USEPA rulemakings to represent that level for long-term exposure (USEPA, 1994c). Similarly, the RfC or surrogate values were used for this analysis. These concepts have also been used as "target cancer risk and target hazard quotients" in a methodology developed for prioritizing environmental problems (Smith, 1996) and for derivation of de minimis levels (USEPA, 1994c) Finally, the level chosen to represent an ample margin of safety for HAPs of concern for non-cancer hazard from short-term exposure was based on Levels of Concern (LOC) developed for the Superfund program (USEPA, 1987, 1994b,c).

Assessing Cancer Hazard

Pollutants were designated as having a "carcinogenic effect" based on the 1986 USEPA Guidelines for Carcinogenic Risk Assessment (USEPA, 1986a). The USEPA proposed revisions to the guidelines in 1996, but the proposed guidelines have not yet been finalized or implemented. Therefore, as a practical matter the weight of evidence and dose-response information used in this analysis relied on assessments using the 1986 guidelines. Accordingly, pollutants described using those guidelines as either Group A (known), B (probable) or C (possible) human carcinogens and any attendant dose-response information for carcinogenicity were used in the determination of benchmark concentrations for cancer concern. For pollutants that do not have USEPA classifications, weight of evidence determinations for carcinogenicity developed by the International Agency for Research on Cancer (IARC) were used. Pollutants are categorized by IARC as Group 1 (agents carcinogenic to humans), Group 2A (probable human carcinogen), and Group 2B (possible human carcinogen). Data considered to be sufficient for classification of HAPs as either Group A, B, or, C (USEPA classifications), or Group 1, 2A, or 2B (IARC classifications) were considered sufficient to identify a HAP as a likely human cancer hazard. Finally, HAPs which have recently had a National Toxicology Program (NTP) study indicating a clear carcinogenic response in animals but which have not been classified by USEPA or IARC, were considered to be potential human carcinogens for this analysis (National Toxicology Program, 1997). However, these HAPs have the greatest uncertainty in assessment of potential cancer hazard.

Sources of data to support benchmark concentrations, carcinogenic weight of evidence determinations, and the prioritization of sources of information were similar to that used for the previous rulemakings (i.e., USEPA's Integrated Risk Information System, USEPA Office of Research and Development documents and assessments, and IARC documents) with the addition of documents from California Environmental Protection Agency's Hot Spots Program and the National Toxicology Program (USEPA, 1994a,b,c; National Toxicology Program, 1997; Risk Assessment Advisory Committee, 1996; Office of Environmental Health Assessment, 1997). The concentration which represents the 1 in a million risk level was derived by dividing the 1×10^{-6} risk level by the inhalation unit risk estimate (in $\mu g/m^3)^{-1}$.

Although USEPA-derived inhalation unit risk (IUR) values were preferred, several potentially carcinogenic HAP do not have these values. When available, other data were used as a surrogate for the IUR. First, surrogates were developed from USEPA-derived oral unit risks expressed in inhalation units (USEPA, 1994a,b). Greater uncertainty is introduced by using such values but they were used here as a better indicator of cancer potency than either a default value or an assumption of no cancer hazard. In addition, USEPA's ED_{10}s (Effective Dose for a 10 percent cancer response above background) were used to derive inhalation unit risks when other values were not available (USEPA, 1994a,b).

When no USEPA values or USEPA-derived surrogates were available, values developed by the California Environmental Protection Agency (CAL/EPA) were used to supplement the USEPA potency estimates. As part of the observations, findings, and recommendations of the CAL/EPA

Risk Assessment Advisory Committee, a comparison of USEPA and CAL/EPA risk assessment practices was conducted with the committee concluding that in general there was good agreement between the two agencies' sets of cancer potency values (Risk Assessment Advisory Committee, 1996). For the most part, values for noncancer and cancer endpoints did not vary by more that a factor of 5. Thus, CAL/EPA carcinogenic potency estimates were considered to be a reasonable surrogate when USEPA values were unavailable.

For HAPs without an USEPA or CAL/EPA potency estimate but with an USEPA or IARC weight of evidence indicating a potential cancer hazard or clear evidence of carcinogenicity in animals in a NTP study, treatment was more qualitative. For such HAPs a default potency value equal to that of methylene chloride—the lowest of the 82 available carcinogenic USEPA-derived potency values for individual HAPs—was assigned. It is unlikely that those HAPs without potency estimates will be lower in potency than the default value and therefore its use is likely to underestimate the hazard.

Assessing Non-cancer Hazard From Long-term Exposure

Toxic endpoints other than cancer and gene mutation are referred to as "non-cancer toxicity" (USEPA, 1990). For derivation of the benchmark concentration for non-cancer hazard from lifetime exposure, USEPA's RfC was used. The RfC is defined as an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime (USEPA, 1990). The RfC is derived from scientific information and science policy and can not be used to quantitatively estimate risk. The RfC methodology is based on the assumption that if the dose to an animal is below that producing the critical toxic effect to the target organ, then all toxic effects are avoided (USEPA, 1994b,c). RfCs used in this analysis have all gone through USEPA agency-wide review, with the exception of one provisional value derived by the Office of Air Quality Planning and Standards and three values that were reported from recent ORD assessments but are in the process of verification (USEPA, 1997).

In the absence of an EPA-derived RfC, Reference Exposure Levels (RELs) for long-term inhalation toxicity —values analogous to RfCs and used in the California Air Toxics "Hot Spot" Program —were used as a surrogate. Again, after comparing USEPA and CAL/EPA noncancer values for long-term exposure, the CAL/EPA Risk Assessment Advisory Committee concluded there was general agreement in the values (Risk Assessment Advisory Committee, 1996).

When either USEPA-derived RfCs or CAL/EPA RELs were unavailable, Minimal Risk Levels (MRLs) developed by the Agency for Toxic Substances and Disease Registry (ATSDR) were used as a surrogate for the RfC. MRLs were developed using a similar methodology to that of the RfC in response to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and represent the maximum exposure levels that would not lead to the development of non-cancer health effects in humans from acute, sub-chronic, and chronic exposures (Agency for Toxic Substances and Disease Registry, 1996).

Assessing Non-Cancer Hazard From Short-Term Exposure

HAPs may be of concern for toxicity from short-term as well as long-term exposures. Comparison of annual average outdoor concentrations to benchmark concentrations intended for short-term exposure can provide an indication of concern for potential health effects because short-term peak concentrations will be higher than annual average concentrations. Ideally, benchmark concentrations for short-term exposure should be based on values such as an RfC for short-term exposure. However to date, there is only one such value that USEPA has developed (ie. RfC_{dt} for the developmental toxicity of ethylene oxide) (USEPA, 1994b, c). EPA has developed an interim approach based upon Levels of Concern (LOCs) established for chemicals on the Superfund Amendments and Reauthorization Act (SARA) Title III section 302 list of "extremely hazardous substances" (USEPA, 1994c).

The LOCs indicate levels of airborne concentrations of chemicals for which no serious irreversible health effects are expected to occur following a short-term exposure of 30 minutes. LOCs are by definition one-tenth of "Immediately Dangerous to Life and Health" levels (IDLHs) developed by National Institute for Occupational Safety and Health (USEPA, 1987). LOCs are the only values used by the USEPA to date which have an extensive database for the HAPs and are designed to protect from serious effects of short-term or acute exposure. However, there are several disadvantages in using LOCs. Since the LOC is based on lethality, the types of health effects represented by extrapolation below that endpoint are not known. Second, the safety factor of 10, applied to IDLHs to protect against serious health effects, may not be adequate. Third, the level of scientific peer review of the rationale for each LOC and supporting data is not as rigorous as for other Agency-derived values such as the RfC. Finally, it is not known what the maximum duration of exposure at the LOC would be for protection against adverse effects (USEPA, 1994c).

Despite these disadvantages, LOCs were used as a basis for determination of benchmark concentrations for 15 HAPs identified as "high concern" for short-term exposure here as well as in USEPA's interim approach (USEPA, 1994b,c). Without the use of such values, the implicit assumption for these HAPs is that there is no concern from short-term exposure, which is inconsistent with the known toxicity of the compounds. To derive a benchmark concentration, the LOC was divided by a safety factor of 1000. This factor was suggested as an appropriate crude estimate of the factor needed to convert the LOC, based upon mortality or very serious effects, into a level that would ensure that no adverse health effects would be observed and to address the concerns listed above (USEPA, 1994c).

Qualitative Considerations

For many HAPs, potential threats to human health have been identified that are not captured by any of the quantitative benchmarks identified above. In the absence of quantitative information, it is important to note those HAPs with significant concerns based on qualitative information. These HAPs include those pollutants identified in the "Great Waters" report to Congress [per section 112(m) of the Clean Air Act] as being of concern not only for toxicity but bioaccumulation and bioconcentration (USEPA, 1994e). These HAPs are deposited to a large extent in large bodies of water from air emissions and pose risks to sensitive ecosystems (USEPA, 1994e). They can

also pose a significant risk to human health through dietary exposure from uptake through the food chain. Accordingly, these pollutants were treated in a qualitative fashion and identified for special concern in this analysis.

In addition, specific HAPs were assigned to a "high-concern" category for severe hazard from short- or long-term exposure as a qualitative indication of hazard in previous USEPA documents (USEPA, 1994b,c). Such HAPs were identified using either LOC or by Composite Score (USEPA, 1994b, c). Composite Scores were originally developed by USEPA for the determination of relative hazard to human health in the Reportable Quantities methodology under the CERCLA (USEPA, 1986b). Composite scores greater than 20 were used to identify HAPs as high concern for hazard from long-term exposure (USEPA, 1994b,c). Accordingly, high-concern HAPs were noted in this analysis as a qualitative indication of hazard.

As an example of the limitations of the existing database available to support quantitative and qualitative determinations of hazard, a large number of HAPs in this analysis (approximately two thirds) have experimental evidence of reproductive and developmental toxicity that ranges widely in quality and in severity of endpoints measured (USEPA, 1993b, 1994d). However, it is beyond the scope of this paper to assign descriptors to specific HAPs that may be of concern for these effects.

Consideration must be given to HAPs which are chemical groups rather than specific compounds. Information concerning chemical groups is shown in Table 1. The table contains hazard information and descriptions of the constituents of outdoor concentration estimates for constituents of chemical groups. In some cases, hazard information can be applied to all members of the chemical class, while in others it can be applied only to individual constituents of the class. In this analysis, when there were adequate data to suggest that the constituents of a chemical class are similar in toxicity, then a single benchmark concentration was assigned to the group as a whole. If evidence was not adequate for such a determination, then the toxicity of individual members of a chemical grouping were assessed and noted independently. This analysis relied primarily on determinations by USEPA for such judgments. For example, the USEPA considers that the carcinogenic hazard to be similar for all cadmium compounds (USEPA, 1994b,c). On the other hand, the hazard of individual members of the glycol ether chemical class varies with each substance and was therefore considered separately. Such considerations are critical for appropriate assignment of benchmark concentrations to HAPs which are chemical classes and for prevention of under- or over-estimation of the health risk.

Tiering of Hazard Information

For the development of a data set of benchmark concentrations for use in identification of potential hazard, all available information needs to be collected from a variety of regulatory sources. However, the need for comprehensive health information must be balanced by accounting for differences in methodology, data, use of uncertainty in derivation, and level of peer review. To address this issue, hazard information was grouped into three tiers as shown in Table 2. Tier I values represent those EPA-derived values with the lowest uncertainties, most comprehensive peer review, and greatest consistency in derivation. Tier II includes other categories of USEPA

TABLE 1. Treatment of Chemical Groups and Their Hazard Information

Compound Class (maximum modeled census tract concentration in µg/m^3)	Compounds with Benchmark Concentrations or Other Information	Benchmark Concentrations in µg/m^3 (Value Type)*	Comments
Antimony (0.12)	Antimony trioxide	0.2 (Chronic - I) 2.0 (Cancer - III) (WOE, B for cancer)	Available information on constituents of modeled antimony compound concentrations is insufficient for application of benchmark concentrations for all antimony compounds.
	Antimony pentafluoride	2.7 (Acute - II)**	
	Antimony potassium tartrate	**	
	Antimony trisulfide	**	
Arsenic (0.28)	All Arsenic compounds	0.00023 (Cancer - I) (WOE, A for Cancer) 0.5 (Chronic- II)	It is appropriate to apply a single cancer and non-cancer potency estimate for long-term exposure to all arsenic compounds. However, available information on constituents of modeled arsenic compound concentrations is insufficient for application of benchmark concentrations for non-cancer toxicity from short-term exposure to all arsenic compounds.
	Arsenic pentoxide	8.0 (Acute - II) **	
	Arsenous oxide	1.4 (Acute - II) **	
	Arsine	1.9 (Acute - II) **	
Beryllium (0.0022)	Metal, oxide, total Beryllium	0.00042 (Cancer-I) (WOE, B2 for cancer) 0.0048 (Chronic - II)	Cancer and non-cancer benchmark concentrations of metal and total beryllium compounds for long-term exposure can be applied to all modeled outdoor concentrations of beryllium compounds. However, available information on constituents of modeled beryllium compound concentrations is insufficient for application of benchmark concentrations of soluble beryllium salts whose presence increases the cancer potency of the emissions.
	Soluble Beryllium Salts (e.g, Beryllium fluoride, Beryllium chloride, Beryllium nitrate, Beryllium phosphate, Beryl ore, Zinc beryllium silicate, Beryllium sulfate)	0.0000012 (Cancer- II) (WOE, B2 for cancer)	

TABLE 1. Treatment of Chemical Groups and Their Hazard Information (cont'd)

Cadmium (0.26)	All compounds	0.00056 (Cancer - I) (WOE, B1 for cancer) 3.5 (Chronic - II) GWP	Cancer and non-cancer benchmark concentrations for long-term exposure can be applied to all modeled outdoor concentrations of cadmium compounds. However, available information on constituents of modeled cadmium compound concentrations is insufficient for application of benchmark concentration for acute toxicity to all cadmium compounds. ***
	Cadmium oxide	4.0 (Acute I) **	
Chromium (1.0)	Total Chromium (based on III and VI valence state)	0.000083 (Cancer - I) (WOE, A for cancer) 0.002 (Chronic - II) 0.05 (Acute - II)	Cancer and non-cancer benchmark concentrations for long-term exposure may be applied to modeled outdoor concentrations of total chromium compounds. The benchmark concentration for non-cancer toxicity from short-term exposure of chromic chloride can also be applied to all modeled outdoor concentrations.
	Chromic chloride (III)	0.05 (Acute - II) **	
Cobalt (0.02)	All Cobalt compounds	0.0050 (Chronic - II) **	Available information on constituents of modeled cobalt compound concentrations is insufficient for application of benchmark concentrations for all cobalt compounds.
	Cobalt Carbonyl	0.27 (Acute - II) **	
	Fluomine	3.0 (Acute - II) **	
Cyanide (1.1)	Hydrogen cyanide	3.0 (Chronic - I)	Available information is insufficient to estimate proportions of these two compounds in modeled concentrations and to therefore apply benchmark concentrations.
	K+ and Na++ Cyanide	5.0 (Acute - II) **	

TABLE 1. Treatment of Chemical Groups and Their Hazard Information (cont'd)

Glycol ethers (24)	2-Ethoxyethanol	20 (Chronic - I)	Available information on constituents of modeled glycol ether compounds is insufficient for application of benchmark concentrations for all glycol ethers.
	2-Methoxyethanol	200 (Chronic - I)	
	Ethylene glycol butyl ether	20 (Chronic - II)	
	Ethylene glycol ethyl acetate	64 (Chronic - II)	
	Ethylene glycol methyl ether acetate	57 (Chronic - II)	
Lead (5.5)	All Lead compounds	(WOE, B2 for Cancer)	The USEPA weight of evidence is for carcinogenicity can be applied to all lead compounds. Similarly the potency estimate for lead acetate can be used as a proxy for the potency of all lead compounds. However there is great uncertainty in assignment of this potency value to the whole group. While there is no evidence to rule out any form of lead as a potential carcinogen, a variety of factors are involved in the mechanisms of lead induced cancer. The non-cancer potency estimate for long-term exposure can be applied to all modeled concentrations. It must be noted that there may be no threshold for non-cancer effects for lead (USEPA, 1994b) and that the primary concern for CAL/EPA site-specific assessments has been non-cancer impacts of lead.***
		0.013 (Cancer - II)	
		1.5 (Chronic - II) **	
		GWP	
	Lead acetate	0.013 (Cancer - II)	
	Tetraethyllead	4.0 (Acute - II) **	
	Tetramethyllead	4.0 (Acute - II) **	

TABLE 1. Treatment of Chemical Groups and Their Hazard Information (cont'd)

Manganese (0.82)	All compounds	0.05 (Chronic - I) **	The non-cancer potency estimate for long-term exposure can be applied to all manganese compounds. However available information is insufficient for application of the benchmark concentration for short-term exposure of methyl cyclo pentadienyl manganese to modeled manganese compound concentrations.
	Methyl cyclo pentadienyl manganese	0.6 (Acute I) **	
Mercury (0.028)	All Mercury compounds	GWP **	Available information on constituents of modeled mercury compounds concentrations is insufficient for application of benchmark concentrations for all mercury compounds. ***
	Mercuric chloride	2.0 (Cancer- III) (WOE, C for cancer)	
	Elemental Mercury	0.3 (Chronic I)	
	Mercuric nitrate	**	
	Mercury (aceto) phenyl	**	
	Methyl mercury	1.0 (Chronic - II)	
Nickel (0.78)	All Nickel compounds	0.0042 (Cancer - I) (WOE, IARC group I)	Potency and weight of evidence for cancer can be assigned to all nickel compounds. IARC assigns a weight of evidence to all nickel compounds as a known carcinogen. CAL/EPA assigns the potency of nickel refinery dust to all nickel compounds and assigns a non-cancer potency estimate for long-term exposure to all compounds. Available information is insufficient for application of the benchmark concentration for short-term exposure of specific nickel compounds to all nickel compounds.
		0.24 (Chronic - II)	
	Nickel refinery dust	0.0042 (Cancer - I) (WOE, A for cancer)	
	Nickel subsulfide	0.0021 (Cancer - I) (WOE, A for cancer)	
	Nickel salts and metal	2.0 (Cancer- III) (WOE, C for cancer)	
	Nickel carbonyl	0.35 (Acute - II) ** (WOE, B for cancer)	

TABLE 1. Treatment of Chemical Groups and Their Hazard Information (cont'd)

		(WOE, B for cancer)	
Polycyclic Organic Matter (POM)	All POM Polycyclic Aromatic Hydrocarbons (PAH)	GWP	Emissions and concentrations estimates are dominated by on Polycyclic Aromatic Hydrocarbons (PAHs)
	Benzo[a]anthracene	0.0048 (WOE, B2 for cancer)	although POM is a category that encompasses thousands of chemicals. Not all PAHs
	Benzo[b]fluoranthene	0.0048 (WOE, B2 for cancer)	are carcinogenic which is why some of those more commonly emitted but with
	Benzo[k]fluoranthene	0.048 (WOE, B2 for cancer)	no evidence of carcinogenicity are included here. Available information
	Benzo[a]pyrene	0.00048 (Cancer - II) (WOE,B2 for cancer)	is insufficient for application of benchmark concentration of specific PAHs to all POM concentrations. The potency
	Dibenzo[ah]anthracene	0.00048 (WOE, B2 for cancer)	estimate of each PAH, expressed in terms that of benzo (a) pyrene, represents
	Indeno[1,2,3-cd]pyrene	0.0048 (WOE, B2 for cancer)	an interim methodology until more research and development of such values
	Anthracene	No evidence of carcinogenic activity	occurs (USEPA, 1993a).
	Phenanthrene	No evidence of carcinogenic activity	
	Dimethylanthracene	No evidence of carcinogenic activity	
	Fluorene	No evidence of carcinogenic activity	
	Fluoranthene	No evidence of carcinogenic activity	
Selenium (0.56)	Selenium compounds	0.5 (Chronic - II) **	The non-cancer potency estimate for long-term exposure can be applied to all
	Selenium sulfide	2.0 (Cancer- III) (WOE, C for cancer)	selenium compounds. Available information on constituents of modeled selenium compounds
	Selenium disulfide	2.0 (Cancer - III) (WOE, C for cancer)	concentrations is insufficient for application of benchmark concentrations for short-term exposure to all selenium
	Sodium selenate	2.3 (Acute - II) **	compounds. Care should be taken in assessment of hazard of selenium compounds for
	Sodium selenite	1.6 (Acute - II) **	while it is quite toxic it is an essential element required by the human body.
	Hydrogen selenide	0.66 (Acute - II) **	

TABLE 1. Treatment of Chemical Groups and Their Hazard Information (cont'd)

[*] Benchmark Concentrations presented for cancer represent a 1/million excess cancer risk. Acute benchmark concentrations are given for the protection of non-cancer effects from short-term exposures. Chronic benchmark concentrations are given for protection of non-cancer effects from long-term exposure.

The notation of GWP describes HAPs of greatest concern under the Great Waters Program (USEPA, 1994c,e). USEPA weight of evidence for carcinogenicity in humans is described as Group A (known human carcinogen), B (probable human carcinogen), and C (possible human carcinogen). That of IARC is group 1 (carcinogenic to humans) group 2A (probably carcinogenic to humans), group 2B (possibly carcinogenic to humans).

[**] "High Concern Pollutants" for either short- or long-term exposure in the section 112(g) hazard ranking (USEPA, 1994b,c)

[***] Members of this chemical group have a tendency for accumulation and concentration in the environment and non-inhalation pathways contribute to its potential toxicity.

data, as well as quantitative information from CAL/EPA and ATSDR. Tier III is comprised mostly of qualitative indicators of potential HAP hazards with the addition of default values and represents the highest degree of quantitative uncertainty of health hazard information used in this analysis.

Comparison with Maximum Modeled Concentrations

The maximum concentration of each HAP estimated by census tract-level modeling of outdoor air toxics concentrations was compiled as part of the Cumulative Exposure Project for comparison with benchmark concentrations. Development of the modeled HAP concentrations for the 60,803 census tracts in the continental United States is described elsewhere (Rosenbaum et al., 1998, and Woodruff et al., 1997, 1998 in press). Emissions data are from mobile and stationary sources for modeled HAPs. The "maximum" concentration used in this analysis was the highest modeled concentration found in any individual census tract across the entire set of census tracts for each HAP. The identification of HAPs whose modeled concentrations exceed benchmark concentrations can provide an indication of HAPs that may be a public health concern.

RESULTS AND DISCUSSION

This analysis has compiled benchmark concentrations and qualitative information describing the potential health hazards posed by 148 hazardous air pollutants and assigned them to tiers as described above. These values, as well as maximum ambient concentrations in air for any individual census tract, are given in Table 3 for 110 specific HAPs with at least quantitative Tier I, II, or III hazard information. As shown in the Table 3, there were 69 Tier I, 98 Tier II, and 13 Tier III values. Chemical groupings were treated separately in Table 1. HAPs without Tier I, II, or III quantitative information are presented in Table 4. HAPs in Table 4 which were considered to be "unrankable" for relative hazard determinations due to lack of information are also noted (USEPA, 1994b,c). Lack of benchmark concentrations for such HAP precludes quantitative analysis of any potential health effects they may cause.

TABLE 2. Tiering and Order of Preference of Benchmark Concentrations and Information

Endpoint	Tier	Priority *	Data source **	Health Effect Value
Cancer (quantitative)	Tier I	1	EPA	Inhalation Unit Risk
	Tier II	2	EPA	Oral Unit Risk values or ED_{10}s expressed in terms of inhalation unit risk values
	Tier II	3	CAL/EPA***	Inhalation Unit Risk values
	Tier III	4	Default	Default potency estimate based on the potency of methylene chloride
Non-cancer (Chronic) (quantitative)	Tier I	1	USEPA	Inhalation Reference Concentrations
	Tier II	2	USEPA	Provisional Inhalation Reference Concentration
	Tier II	3	CAL/EPA***	Reference Exposure Levels developed for the California Hot Spots Program for chronic toxicity
	Tier II	4	ATSDR	Minimal Risk Levels developed for chronic toxicity
Non-cancer (Acute) (quantitative)	Tier I	1	USEPA	Inhalation Reference Concentration for developmental toxicity (RfCdt)
	Tier II	2	USEPA	Levels of Concern/1000 for protection from short-term exposure for the HAPs identified as being of high concern for acute toxicity under section 112(g)
Other information (qualitative)	Tier I	n/a	USEPA or IARC	Weight of evidence as known, probable, or possible human carcinogen
	Tier III	n/a	NTP	Clear evidence of animal carcinogenicity in an NTP study in the absence of an EPA or IARC determination
	Tier III	n/a	USEPA	Identification of the HAP as being of concern in the Great Waters Report to Congress
	Tier III	n/a	USEPA	Identification of the HAP as a "High-Concern Pollutant" for severe toxicity from relatively low long- or short-term exposure

*Prioritization is the order of preference for different types of information when more than one type was available for a specific tier.

**"USEPA" is the United States Environmental Protection Agency, "ATSDR" is the Agency for Toxic Substances and Disease Registry, "CAL/EPA" is the California Environmental Protection Agency, "IARC" is the International Agency for Research on Cancer, and "NTP" is the National Toxicology Program.

***CAL/EPA has released new draft numbers that have not had peer or public review completed. The new draft numbers will be used as a supplement but not replacement to those values presented in the final Risk Assessment Advisory Committee report (Risk Assessment Advisory Committee, 1996, Office of Environmental Health Assessment, 1997).

These results show that while many of the HAPs have at least one type of Tier I or Tier II benchmark concentration, the number of such benchmarks needed to fully characterize the potential hazard of this group of HAPs is far from complete. Development of benchmarks of similar quality and consistency is needed for a large number of the HAPs. For example, there are 13 specific HAPs and 4 chemical groupings containing members with a weight of evidence of potential carcinogenicity or a recent NTP study reporting clear evidence of animal carcinogenicity but no potency estimate. Similarly, many HAPs do not have benchmark concentrations for non-cancer hazard. While all HAPs should not necessarily have weight of evidence or potency information for cancer, all should have benchmark concentrations identified for non-cancer hazard as all have the potential to be a hazard from this type of toxicity. Further development of quantitative data characterizing cancer and non-cancer risks from HAPs will greatly enhance efforts to evaluate the potential impacts of these pollutants. This is especially true for those HAPs that may be of concern from short-term exposure where the uncertainties involved with use of the LOC/1000 are large.

The hazard information for listed pollutants that are members of a chemical class requires special consideration. The list of hazardous air pollutants contained in section 112 of the Clean Air Act Amendments of 1990 identifies 189 pollutants of which 17 are actually chemical classes (e.g., beryllium compounds). A prominent feature of Table 1 is the lack of benchmark concentrations for a large number of chemicals belonging to groups. Table 1 also illustrates that careful analysis of the constituents of chemical groups and their modeled concentrations are needed so that appropriate benchmark concentrations can be assigned.

This analysis emphasizes the inhalation route of exposure as benchmark concentrations were applied to modeled ambient air concentrations. However, health effects information is not always available for the inhalation route of exposure and extrapolations were needed to use available information from other routes of exposure. When extrapolating between two different routes of exposure (e.g., inhalation vs oral), a number of factors are important for determining the association between a specific dose and the degree of toxic response engendered by a pollutant. These factors include differences by route of exposure in (1) tissue distribution, (2) rate of delivery leading to differing concentration profiles, (3) degree of metabolism, and (4) response caused by an agent at its site of action across species and among target tissue. How such uncertainties affected the application of dose response information for this type of analysis is not clear (USEPA, 1994b). However, in limited comparisons of differences between oral and inhalation dose routes associated with either a 1 percent or 25 percent additional risk of cancer, Pepelko (1990) concluded that the carcinogenic potencies are not substantially influenced by dose route. However, the use of information extrapolated from oral to the inhalation route of exposure involves greater uncertainty than using that based on the inhalation route. This uncertainty is addressed by assignment of benchmark concentrations based on extrapolated data to Tier II rather than Tier I.

TABLE 3. Pollutants Which Have Tier I, II or III Benchmark Concentrations Values for Cancer, and Noncancer Effects from Long-term (Chronic) or Short-term (Acute) Exposure*

Pollutant Name	CAS #	Carcinogenic Weight of Evidence**	One-per million cancer risk ($\mu g/m^3$)	Noncancer (chronic) ($\mu g/m^3$)	Noncancer (acute) ($\mu g/m^3$)	Maximum Modeled Census Tract Concentration ($\mu g/m^3$)
Acetaldehyde	75070	B	0.45 (I)	9.0 (I)	_	21
Acetamide	60355	IARC 2B	0.050 (II)	-	-	6.5 E-6
Acetonitrile	75058	-	-	50 (II)	-	1.8
Acrylamide	79061	B	0.00077 (I)	0.7 (II)	-	0.040
Acrylic acid	79107	-	-	1.0 (I)	-	0.50
Acrylonitrile	107131	B	0.015 (I)	2.0 (I)	-	7.7
Acrolein	107028	C	2.0 (III)	0.020 (I)	1.2 (II) *	20
Allyl chloride	107051	C	0.17 (II)	1.0 (I)	-	0.56
Aniline	62533	B	0.63 (II)	1.0 (I)	-	4.0
Anisidine	90040	IARC 2B	0.025 (II)	-	-	0.0027
Benzene	71432	A	0.12 (I)	71 (II)	-	79
Benzotrichloride	98077	B	0.00028 (II)	-	0.70 (II) *	0.020
Benzyl chloride	100447	B	0.020 (II)	12 (II)	5.2 (II) *	0.26
Bis(chloro-methyl) ether	542881	A	0.000016 (I)	-	-	0.0015
Bromoform	75252	B	0.91 (I)	-	-	1.1
1.3-Butadiene	106990	B	0.0036 (I)	8.0 (II)	-	6.7
Captan	133062	B	1.0 (II)	-	-	0.12
Carbon disulfide	75150	-	-	700 (I) *	-	58
Carbon tetrachloride	56235	B	0.067 (I)	2.4 (II)	-	4.8
Chlordane	57749	B	0.0027 (I)	0.018 (II) GWP	-	0.04
Chloroacetic acid	79118	-	-	-	1.8 (II) *	0.15
Chlorobenzene	108907	-	-	70 (II)	-	38
Chloroform	67663	B	0.043 (I)	35 (II)	-	8.8
Chloromethyl methyl ether	107302	A	0.0014 (II)	-	1.8 (II) *	0.010
Chloroprene	126998	+ in NTP study but not classified	2.0 (III)	1.0 (II)	-	12
Cresols	-	C	2.0 (III)	180 (II)	-	1.7
DEHP (Bis [2-ethylhexyl] phthalate)	117817	B	0.25 (II)	71 (II)	-	2.6
1,4 - Dichlorobenzene(p)	106467	C	0.15 (II)	800 (I)	-	28
3,3-Dichlorobenzidine	91941	B	0.0078 (II)	-	-	0.00073

TABLE 3. Pollutants Which Have Tier I, II or III Benchmark Concentrations Values for Cancer, and Noncancer Effects from Long-term (Chronic) or Short-term (Acute) Exposure[*] (cont'd)

Pollutant Name	CAS #	Carcinogenic Weight of Evidence[**]	One-per million cancer risk ($\mu g/m^3$)	Noncancer (chronic) ($\mu g/m^3$)	Noncancer (acute) ($\mu g/m^3$)	Maximum Modeled Census Tract Concentration ($\mu g/m^3$)
Dichloroethyl ether [bis(2-chloroethyl) ethyl]ether	111444	B	0.0030 (I)	-	-	0.030
1,3, Dichloropropene	542756	B	0.027 (I)	20 (I)	-	1.1
Dichlorvos	62737	B	0.012 (II)	0.50 (I)	-	0.00073
Diethyl sulfate	64675	IARC 2A	2.0 (III)	-	-	0.02
3,3-Dimethoxybenz-idine (dianisidine)	119904	B	0.0067 (II)	-	-	0.000030
Dimethyl formamide	68122	IARC 2B	2.0 (III)	30 (I)	-	5.0
1,1-Dimethyl hydrazine	57147	B	0.00040 (II)	0.022 (II)	-	0.0045
Dimethyl sulfate	131113	B	2.0 (III)	-	5.0 (II) [*]	0.040
4,6-Dinitro-o-cresol and salts	534521	-	-	-	0.5 (II) [*]	0.0018
2,4-Dinitrotoluene	121142	B	0.0091 (II)	7.0 (II)	-	0.06
1,4-Dioxane (1,4-Diethylene oxide)	123911	B	0.32 (II)	400 (II)	-	0.75
Epichlorohydrin	106898	B	0.83 (I)	1 (I)	-	0.41
1,2-Epoxybutane	106887	under review	-	20 (I)	-	0.19
Ethyl acrylate	140885	B	0.073[****] (II)	48 (II)	-	4.4
Ethyl benzene	100414	+ in NTP study but not classified	2.0 (III)	1000 (I)	-	9.0
Ethyl carbamate (Urethane)	51796	B	0.036 (II)	-	-	0.010
Ethyl chloride	75003	+ in NTP study but not classified	2.0 (III)	10,000 (I)	-	10
Ethylene dibromide (Dibromoethane)	106934	B	0.0045 (I)	0.20 (II)	-	1.2
Ethylene dichloride (1,2-Dichloroethane)	107062	B	0.038 (I)	95 (II)	-	22
Ethylene oxide	75218	B	0.043 (II)	600 (II)	540 (I) [*]	1.9
Ethylene thiourea	96457	B	0.032 (II)	3.0 (II)	-	0.0012
Ethylidene dichloride (1,1-Dichloroethane)	75343	C	0.63 (II)	-	-	5.0×10^{-5}
Formaldehyde	50000	B	0.077 (I)	3.6 (II)	-	52

TABLE 3. Pollutants Which Have Tier I, II or III Benchmark Concentrations Values for Cancer, and Noncancer Effects from Long-term (Chronic) or Short-term (Acute) Exposure[*] (cont'd)

Pollutant Name	CAS #	Carcinogenic Weight of Evidence[**]	One-per million cancer risk ($\mu g/m^3$)	Noncancer (chronic) ($\mu g/m^3$)	Noncancer (acute) ($\mu g/m^3$)	Maximum Modeled Census Tract Concentration ($\mu g/m^3$)
Heptachlor	76448	B	0.00077 (I)	-	-	0.060
Hexachlorobenzene	118741	B	0.0022 (I)	2.8 (II) GWP	-	0.060
Hexachlorobutadiene	87633	C	0.045 (I)	90 (II)	-	0.010
Hexachlorocyclopentadiene	77474	-	-	0.070 (II)	0.020 (II) [*]	0.79
Hexachloroethane	67721	C	0.25 (I)	80 (II)	-	0.040
Hexane	110543	-	-	200 (I)	-	72
Hydrazine	302012	B	0.00020 (I)	0.24 (II)	-	0.030
Hydrochloric acid	7647010	-	-	20 (I)	-	300
Hydrogen fluoride	7664393	-	-	5.9 (II)	1.6 (II) [*]	2.2
Lindane	58899	B/C	0.0026 (I)	1.0 (II) GWP	-	0.010
Maleic anhydride	108316	-	-	2.4 (II) [*]	-	2.4
Methanol	67561	-	-	620 (II)	-	63
Methyl bromide (Bromomethane)	74839	-	-	5.0 (I) [*]	-	1.5
Methyl chloride (Chloromethane)	74873	C	0.56 (I)	-	-	7.9
Methyl chloroform (1,1,1-Trichloroethane)	71556	-	-	320 (II)	-	40
Methyl ethyl ketone	78933	-	-	1000 (I)	-	87
Methyl hydrazine	60344	B	0.0032 (II)	-	0.94 (II) [*]	0.010
Methyl iodide	74884	C	2.0 (III)	10 (I)	-	0.14
Methyl isocyanate	624839	-	-	0.36 (II)	4.7 (II) [*]	0.11
Methyl methacrylate	80626	-	-	980 (II)	-	37
Methyl tert-butyl ether	1634044	B/C	6 .0 [***] (II)	3000 (I)	-	40
4,4-Methylenedianiline	101779	IARC 2B	0.0022 (II)	1.9 (II)	-	0.020
4,4-Methylene bis (2-chloroaniline)	101144	B	0.011 (II)	-	-	0.010
Methylene chloride (Dichloromethane)	75092	B	2.1 (I)	3000 (II)	-	51
Methylene diphenyl diisocyanate	101688	-	-	0.020 (I) [*]	-	2.3

TABLE 3. Pollutants Which Have Tier I, II or III Benchmark Concentrations Values for Cancer, and Noncancer Effects from Long-term (Chronic) or Short-term (Acute) Exposure* (cont'd)

Pollutant Name	CAS #	Carcinogenic Weight of Evidence**	One-per million cancer risk ($\mu g/m^3$)	Noncancer (chronic) ($\mu g/m^3$)	Noncancer (acute) ($\mu g/m^3$)	Maximum Modeled Census Tract Concentration ($\mu g/m^3$)
Naphthalene	91203	-	-	14 (II)	-	3.9
Nitrobenzene	98953	-	-	1.7 (II) *	-	0.26
2-Nitropropane	79469	B	2.0 (III)	20 (I)	-	0.040
Parathion	56382	C	2.0 (III)	-	2.0 (II) *	0.010
Pentachloronitrobe-nzene	82688	C	0.014 (II)	-	-	0.0019
Pentachlorophenol	87865	B	0.033 (II)	0.20 (II)	-	0.01
Phthalic anhydride	85449	-	-	120 (I)	-	7.4
Phenol	108952	-	-	45 (II) *	-	38
Phosgene	75445	-	-	0.30 (II)	0.80 (II) *	0.10
Polychlorinated Biphenyls (Arochlors)	1336363	B	0.0020 (I) Highest	1.2 (II) GWP	-	0.020
Propoxur	114261	B	0.91 (II)	-	-	0.000048
Propylene dichloride (1,2-Dichloropropane)	78875	B	0.053 (II)	4 (I)	-	3.4
Propylene oxide	75569	B	0.27 (I)	30 (I)	-	1.3
1,2-Propylenimine	75558	B	0.00015 (II)	-	-	0.010
Quinoline	91225	C	0.00029 (II)	-	-	0.030
Styrene	100425	C	2.0 (III)	1000 (I)	-	31
Styrene oxide	96093	IARC 2A	0.022 (II)	6.0 (II)	-	0.0038
2,3,7,8 - Tetrachloro-dibenzo-p-dioxin (Dioxin)	1746016	B	3.0E-8 (I)	0.0000035 (II)	-	6.1E-7
1,1,2,2-Tetrachloro-ethane	79345	C	0.017 (I)	-	-	0.080
Tetrachloroethylene (Perchloroethylene)	127184	B/C	1.7 (I)	35 (II)	-	39
Toluene	108883	-	-	400 (I)	-	89
2,4-Toluene diamine	95807	B	0.0011 (II)	-	-	0.010
2,4-Toulene diisocyanate	584849	IARC 2B	0.091 (II)	0.07 (I)	7.0 (II)*	0.26
o-Toluidine	95534	B	0.18 (II)	-	-	0.010
1,2,4-Trichlorobenzene	120821	-	-	200 (I)	-	0.35

TABLE 3. Pollutants Which Have Tier I, II or III Benchmark Concentrations Values for Cancer, and Noncancer Effects from Long-term (Chronic) or Short-term (Acute) Exposure[*] (cont'd)

Pollutant Name	CAS #	Carcinogenic Weight of Evidence[**]	One-per million cancer risk ($\mu g/m^3$)	Noncancer (chronic) ($\mu g/m^3$)	Noncancer (acute) ($\mu g/m^3$)	Maximum Modeled Census Tract Concentration ($\mu g/m^3$)
1,1,2-Trichloroethane	79005	C	0.063 (I)	400 (II)	-	4.3
Trichloroethylene	79016	B\C	0.59 (I)	640 (II)	-	32
2,4,6-Trichlorophenol	88062	B	0.32 (I)	-	-	0.00040
Trifluralin	1582098	C	0.45 (II)	-	-	0.04
Vinyl acetate	108054	C	2.0 (III)	200 (I)	-	20
Vinyl bromide (Bromomethane)	593602	B	0.031 (I)	3 (I)	-	0.010
Vinyl chloride	75014	A	0.012 (I)	26 (II)	-	13
Vinylidene chloride (1,1-Dichloroethylene)	75354	C	0.020 (I)	32 (II)	-	0.51
Xylenes	1330207	-	-	300 (II)	-	72

[*] HCP notes pollutants assigned to the "high-concern" category for severe hazard from short- or long-term exposures (USEPA, 1994b,c).

[**] USEPA weight of evidence for carcinogenicity in humans is described as Group A (known human carcinogen), B (probable human carcinogen), and C (possible human carcinogen). That of IARC is group 1 (carcinogenic to humans) group 2A (probably carcinogenic to humans), and group 2B (possibly carcinogenic to humans).

[***] This determination is from an USEPA draft document for the assessment of hazards of MTBE (National Science and Technology Council, 1996).

[****] The actual inhalation risk may be lower for this HAP as the cancer risk value is based on oral data with apparent portal of entry effects (Peer Reveiw Panel, 1995).

[*****] USEPA weight of evidence is different that that in the technical support document for section 112(g) (USEPA, 1988a).

TABLE 4. Pollutants Without Tier I, II, or III Quantitative Information

Pollutant Name	CAS No.	Carcinogenic WOE	Composite Score (CS), other non-cancer descriptors ***	Maximum Modeled Census Tract Concentration ($\mu g/m^3$)
Acetophenone	98862	-	CS>20 *	0.00045
Biphenyl	92524	-	CS <20	1.3
Calcium cyanamide	156627	-	CS <20	0.03
Carbaryl	63252	-	CS <20	0.020
Carbonyl sulfide	463581	-	Unrankable	28
Catechol	120809	IARC 3	Unrankable	0.15
Chloramben	133904	WOE UR**	-	1.4 E-5
Cumene	98828	-	CS <20	8.6
2,4,-D, Salts and esters (2,4-Diclorophenoxy acetic acid)	94757	-	CS <20	0.03
Dibutylphthalate	84742	-	CS<20	0.13
Diethanolamine	11422	-	unrankable	0.09
N,N - Dimethylaniline	121697	-	CS>20 *	0.12
Dimethyl phthalate	131113	-	CS<20	0.49
2,4-Dinitrophenol	51285	-	CS>20 *	0.0034
Ethylene glycol	107211	-	CS<20	38
Hydroquinone	123319	-	CS<20	0.04
Methoxychlor	72435	-	CS<20	0.01
Methyl isobutyl ketone	108101	-	CS<20	9.9
4 - Nitrophenol	100027	-	unrankable	0.03
p-Phenylenediamine	106503	-	CS<20	0.0014
Propionaldehyde	123386	-	unrankable	4.0
Quinone	106514	IARC 3	unrankable	0.010
2,2,4-Trimethylpentane	540841	-	unrankable	23

*"HCP" notes High Concern Pollutant and "unrankable" notes HAPs with insufficient information to be relatively ranked in the section 112(g) hazard ranking (USEPA, 1994b,c).
**WOE UR notes weight of evidence under review.
***Composite Scores greater than 20 are used to identify HAPs as being of "High Concern" for non-cancer toxicity from long-term exposure (USEPA, 1994b,c).

TABLE 5. Number and Type of Benchmark Concentrations and Number of Exceedances of them by Maximum Outdoor Concentration*

TIER	Type of Toxicity	Number of benchmark concentrations for HAPs *	Number of Exceedances of benchmark by maximum modeled outdoor concentration *
Tier I	Cancer	40	32
	Non-cancer (long-term exposure)	33	8
	Non-cancer (short-term exposure)	1	0
Tier II	Cancer	37	25
	Non-cancer (long-term exposure)	57	15
	Non-cancer (short-term exposure)	15	3
Tier III	Cancer	14	7

* Included are specific HAPs as well as chemical groupings which all members of the group may be treated as a single compound for assignment benchmark concentrations for cancer or non-cancer effects.

There are uncertainties in addition to those incurred through the use of oral data in the benchmark concentrations presented here. The benchmark concentration for cancer hazard is derived from the unit risk, an upper-bound estimate of the excess cancer risk over background associated with a continuous lifetime exposure. There are many uncertainties associated with inferences of population risk based on upon the estimate of the unit risk. Factors including use of sensitive animal strains, tumor sites of uncertain human relevance, and linear extrapolation to low doses can contribute to uncertainty in estimating the risk in the human population (Cogliano, 1997). Differences in the quality of data for any one pollutant varies and can be expressed as a factor in the weight of evidence determination for cancer hazard in humans. Differences in the pharmacokinetics of a pollutant is expected between exposure routes and species and can have influence on extrapolation of observed responses in animals and humans (USEPA, 1994b).

As with the unit risk, the RfC contains uncertainty and by definition is an estimate with an uncertainty spanning perhaps an order of magnitude (USEPA, 1994b). The RfC is limited in its consideration of severity of effect and although there is an application of severity in the RfC methodology it does not include a numerical adjustment. However, considerations of uncertainty are numerically represented in the derivation of RfC to account for differences in human sensitivity, extrapolation from animals to humans, length of study, use of an observed rather than non-observed effect level, and completeness of the database describing the pollutant. The greater the uncertainty in non-cancer hazard information for a pollutant, the greater the use of conservative safety factors in derivation of the RfC. However if the uncertainty is too great it is determined that data is insufficient for derivation of an RfC. (USEPA, 1990)

The RfC as a measure of noncancer toxicity has less uncertainty as an indicator of possible health effects by inhalation exposure in comparison with methods dependent on derivation of inhalation hazard from oral data. Dosimetric adjustments to account for the species-specific relationships of inhaled concentrations and deposited/ delivered doses, separate treament of gases and particles, and the site of the observed toxic effect (respiratory or extrarespiratory) are all considered in the derivation of the RfC (USEPA, 1990).

For non-cancer values, in the absence of an RfC, an option was to use the Reference Dose (RfD) for determination of non-cancer hazard of the pollutant (an RfD is similar to an RfC except it is an estimate for oral exposure). However, oral studies are limited as indicators of non-cancer inhalation toxicity because of factors such as portal of entry effects and liver "first-pass effects" as well as lack of consideration of dosimetric considerations (USEPA, 1994b). Accordingly, inhalation values for non-cancer health effects were relied upon to describe such hazard rather than oral values such as the RfD. Alternate values including CAL/EPA RELs and ATSDR MRLs were used as benchmark concentrations for non-cancer health effects when no RfC was available.

The number of benchmark concentrations, for specific HAPs or for chemical groupings for which a benchmark concentration can be applied to all members of the group, exceeded by modeled estimates of the maximum ambient concentration of HAPs across all census tracts is shown in Table 5. Out of a total of 197 benchmark concentrations presented, approximately 45 percent were exceeded. However, there was not a equal distribution of exceedances across the different types of benchmarks with approximately 70 percent of cancer and 25 percent of noncancer benchmarks exceeded. The distribution of exceedances was also not uniform across tier with approximately 50 percent of Tier I and 40 percent of Tier II values exceeded.

These "maximum" modeled concentrations are not meant to represent the highest concentration which would ever occur in the U.S.: variations in concentrations within census tracts, in particular at locations nearest to large emissions sources, may produce greater concentrations than those estimated as average census tract values. In addition, short-term peak concentrations will be greater than long-term average concentrations.

The maximum census tract values produced by the model, however, have high uncertainties. For several compounds, the maximum concentration estimate exceeds any values reported in previous monitoring studies. In many cases, these high concentrations are derived from high emissions estimates which may be overestimates. Future work will further evaluate these high concentrations. However, for many compounds the maximum model value exceeds the benchmark concentration identified in this paper by a large margin. In such cases, even if the maximum value is an overestimate, it is still very likely that at least one census tract has a concentration in excess of the benchmark.

There are additional issues that add uncertainty to conclusions drawn from comparison of health information assembled in this study with estimates of ambient concentrations. Multimedia contributions from food and water intake were also not taken into account in this analysis. This may lead to significant underestimation of the hazard for HAPs such as mercury where other

routes of exposure are important. In addition, the aggregate hazard of exposure to multiple HAPs in an individual census tract was not addressed in this analysis. Due to the paucity of health effects data for most of the HAPs, there may not be an awareness of potential health effects because they have not been described. Finally, the hazard represented by ambient concentrations of a HAP may be underestimated as a relatively less toxic HAP may be transformed in the atmosphere to chemicals not on the HAP list but with greater toxicity (Dumdie et al., 1988; Kelly et al., 1994).

Even though there are many limitations to the available hazard data for HAPs, this examination provides a first step quantitative comparison of estimated maximum ambient concentrations and the existing quantitative information on benchmark concentrations as indicators of potential health hazard. A primary focus of this work is the assemblage of appropriate benchmark concentrations and the attendant science policy considerations. Application to maximum concentrations of each HAP is an initial screen for toxicity but is only a preliminary and limited use of the assemblage and hierarchy of benchmark concentrations presented here. A more complete and comprehensive analysis which includes further comparisons of benchmark concentrations and estimated ambient concentrations by census tract is needed in order to proceed with prioritization of hazard posed by the HAPs and is the subject of a separate analysis (Woodruff et al., 1998).

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